**Herpes Zoster of the Finger.**—SUERMONPREZ and PLATEL (*Journ. des Mal. Cut. et Syph.*, December, 1899) assert that affection of the fingers in this disease is not so rare as is generally supposed. It usually affects the metacarpal or middle phalanges of the fingers, and is very rare on the thumb. The authors believe it to be due, in every instance, to a direct infection of the finger, traumatism being only of secondary importance in its etiology. It differs from herpes elsewhere in the absence in the beginning of local congestion, a large, flat bleb being the first lesion that is noted.

## PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
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**Cirrhosis and Multiple Adenomata of the Liver.**—SCHMIEDER (*Virchow's Archiv*, 1900, vol. clix., p. 290) reports a case of multiple adenomata of the liver with accompanying cirrhosis, and discusses the relation between simple hypertrophy, adenomata, and carcinomata. He also expresses his belief as to the factors giving rise to adenomata of the liver.

In his review of the literature he shows that a sharp line between hyperplasia of hepatic tissue and adenomatous formation cannot be drawn, and that many of the reported cases of adenomata of the liver are combined with cirrhosis of that organ.

Schmieder gives a full clinical history of his case and detailed study of the liver, both macroscopically and microscopically. Careful search reveals to him no connection whatever between the interlobular bile-ducts and the adenomatous formations. On the other hand, he proceeds to demonstrate that the islands of liver cells which are cut off and included by the connective tissue in the cirrhotic process are the points of origin of multiple adenomata. In the centre of these isolated masses of hepatic tissue the liver cells take on a vicarious growth, and one can recognize that the most centrally lying cells are largest. Their structure corresponds completely to normal liver cells, with the exception that they are larger. Single and multinuclear giant cells are found here, and it is by the division of these giant cells that adenomatous cells arise.

His sections of very small adenomata reveal frequently the transition of large multinuclear liver cells into columns of adenomatous cells. Once these columns have arisen the subsequent tumor growth is very rapid, and soon all trace as to its origin is completely lost.

He also finds in isolated patches of large adenomata a change in character of the tumor which approaches cancer. This is shown by thick columns of tumor cells assuming an alveolar arrangement, by the centre of the growth becoming converted into a mass of cells devoid of definite arrangement, and, finally, by the actual demonstration; in a small area, of true cancerous growth.

Schmieder regards the cirrhotic process as preceding the adenomatous formations, and advances in support of his view the fact that cirrhosis is a diffuse condition, occupying all parts of the liver, and is, consequently, found in portions where adenomata do not exist; that his microscopical study convinces him that cirrhosis is an extremely chronic process, while the tumor formation is a recent and rapidly developing one; and, finally, the clinical symptoms pointing to cirrhosis extended in his case over a period of two years.

In conclusion, he emphasizes his conception that cirrhosis is the first factor in the production of adenomata of the liver, and that the establishment of this view would give a means by which the multiple adenomata might be classified in a special group; at the same time he warns against the assumption that the tumor formation in this case may be placed parallel with the origin of hepatic tumors in general.

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**Structure and Origin of the Cavernomata of the Liver.**—SCHMIEDEN (*Virchow's Archiv*, 1900, vol. clxi., p. 373), in a well-written article emanating from Orth's laboratory, discusses at length the five theories so far proposed in regard to the origin of the cavernomata of the liver, and on the basis of the study of thirty-two cases coming under his own observation adds a sixth theory, in favor of which he advances strong arguments based on histological findings and embryological considerations.

Cavernomata of the liver have frequently been the object of investigations ever since they were first described by Virchow. The interest taken in them stands in no relation to their clinical and pathological significance. They are frequently found at autopsies, but have no bearing on the other lesions of the liver or of the body in general. The point in dispute is their histogenesis; the developed tumor possesses little interest.

The five theories of the starting-point of these cavernous formations are the following:

1. Primary growth of connective tissue.
2. Vascular congestion.
3. Primary atrophy of liver cells.
4. Biliary congestion.
5. Hemorrhage.

Of these theories only two deserve serious consideration, namely, the first and second. The other three have little or no evidence in their favor.

The origin from a growth of connective tissue is the oldest theory, and is at present strongly upheld by Ribbert, who uses the liver cavernoma as a model for all cavernous angiomata, and regards it as an angiofibroma. Against this view is the fact that liver angiomata occur with no connective tissue, but with only liver cells between the blood spaces. It may also be asked why the connective tissue takes the well-known form of the liver cavernoma instead of the structure of cirrhosis or of a fibroma, and when the growth of the tumor stops after a certain time?